whom disease was limited, at presentation, to the lingula (5) or middle lobe (1). All were women free of clinically significant PEJD. Caseating granulomata and bronchiectasis were evident in one surgically treated patient; the rest received nonsurgical treatment and their pathologic anatomy is unknown. We speculated that voluntary cough suppression (VCS) resulted in stasis and secondary infection in the dependent tips of these poorly draining lobes, leading to localized bronchiectasis on which pulmonary MAC engrafted. We hypothesized that VCS was due to fastidiousness and suggested the term “Ladys Windermere Syndrome” (LWS) for the entity. Two non-MAC patients, both women, in whom evidence that VCS had induced or modified their disease was indubitable, were cited (in the index section) to support the existence of this mechanism of disease: one had bilobar atelectasis, and the other, predominantly basal tuberculosis.

Middle-aged to elderly women comprised most of the 129 persons with nonobstructive lingular and middle lobe syndromes reported from Duke University.4 Bronchiectasis, organizing pneumonia, and bronchial mucous plugging were the major findings among the 38 persons who underwent surgery. Of 50 patients accrued since publication, 46 (92%) are women (C.E. Putnam, MD, written communication, 1992).

Third, what is the genesis of the cylindrical bronchiectasis encountered predominantly in female patients with diffuse nodular pulmonary MAC?2

Drs. Wallace1 and Swensen et al5 suggest that the micronodular pulmonary MAC may be causal. We think this is unlikely: (1) engraftment of MAC on bronchiectasis conforms with a known sequence; (2) cylindrical bronchiectasis among elderly women was encountered with some frequency in the absence of the micronodular pattern but the reverse circumstance appears to be rare to nonexistent; (3) posttuberculous bronchiectasis is typically a consequence of exudative, often cavitary tuberculosis; this sequela is not known to occur in nonexudative forms, eg, mililiary tuberculosis.

In 1993, Wells et al6 showed that cylindrical bronchiectasis could be a consequence of VCS. They presented the cases of four young women with recurring lower respiratory infections directly attributable to VCS; bilateral cylindrical bronchiectasis was found in two. An air-fluid level in the posterior trachea, evident on computerized tomography, and caused by the presence of copious pooled proximal secretions, was found to be a marker of VCS.

It is clear that (1) VCS can induce a number of pulmonary syndromes-cylindrical bronchiectasis, lobar atelectasis, bibasal tuberculosis, and possibly LWS; (2) this pathogenetic mechanism has been exclusively reported in women; and (3) individuals with “peripheral middle lobe syndrome,” which encompasses disease of either the middle lobe or lingula, are preponderantly women. Is VCS the cause of the propensity exhibited by women to develop bronchiectasis associated with micronodular pulmonary MAC,5 nonobstructive middle lobe syndrome,4 and lingular-middle lobe localization of pulmonary MAC in LWS55 An affirmative answer would provide a unifying explanation for these seemingly unrelated observations.

Is the LWS hypothesis testable? We considered psychometric evaluation of fastidiousness in our small sample but were unable to find a test that measured this trait. A capsaicin cough-provocation challenge would be helpful, provided that cough intensity, rather than threshold, was assessed, eg, by summing cough decibel generation, and suitable controls were available. These would have to include age- and smoking status-matched patients of both sexes, and women with MAC of similar extent but dissimilar distribution, ie, upper lobe vs lingular disease. A review of the Mayo Clinic images7 could be supportive of this hypothesis if air-fluid levels in the trachea were shown in some cases, or if women were found to have predominance of lingula or middle lobe bronchiectatic involvement as compared with men. Incidentally, Figure 1, in the article by Swensen et al,5 shows middle lobe bronchiectasis in a gender-unspecified person.

Respiratory disease caused by VCS, by which we mean obtundation of the cough reflex by habitual suppression, is analogous to other diseases due to disordered function seen preponderantly in women: defecatory reflex and rectal constipation; detrusor reflex and urinary infections; and hunger response and anorexia nervosa. VCS is both difficult to recognize and confirm; it may be a good deal more common than the limited number of reports would suggest. If that is the case, it may play a wider role as a pathogenetic mechanism in pulmonary disease than has previously been recognized.

Jerome M. Reich, MD, FCCP, Pulmonary Division, Bess Kaiser Medical Center, Northwest Permanente, Portland, Oregon; and Richard E. Johnson, PhD, Senior Investigator, Center for Health Research, Kaiser Permanente, Northwest Region Oregon

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To the Editor:

In response to an editorial we had written (Chest 1994; 105:6-7), Drs. Reich and Johnson have provided several concepts to explain the somewhat late recognition of the involvement of women with Mycobacterium avium complex (MAC) lung disease. The first of these deals with the impact of disease definition and types of referral centers on the demographics of the disease. The bias of reporting only patients with moderate to far advanced lung disease from tertiary care centers in most early studies of MAC is understandable given the reluctance of early investigators to accept infection as disease without cavitory or unequivocal radiographic changes compatible with granulomatous disease. Given that most women with this disease have no defined underlying lung disease, other than possibly bronchiectasis which will be addressed later, and tend to have less advanced disease, more men than women would be reported. We agree that the type of disease and the reporting from referral centers may have biased these early studies. Clearly more community based studies, perhaps using two or more positive sputum-bronchoscopy samples as...
the only required diagnostic criteria, would give a better picture of current man to woman representation of this disease.

Reich and Johnson then present their argument that the bronchiectasis seen in most women with MAC disease is a causative factor rather than a consequence of the disease. Unquestionably, there are some women (and men) with lingular and middle lobe syndromes unrelated to MAC disease. The studies by Hartman et al.1 and Swensen et al. (Chest 1994; 105:49-52), which prompted our initial editorial, clearly pointed out the significance of coexisting nodular disease as a major radiographic marker for identifying the patients with bronchiectasis who had MAC disease. More than 50% of patients with bronchiectasis did not have nodular disease and had negative mycobacterial cultures. They presumably have another disease. There are also unquestionably some patients, especially those with prior treated mycobacterial infections, eg, Mycobacterium tuberculosis, or cystic fibrosis, in which the MAC follows in the wake of already established bronchiectatic areas of the lung. In patients with coexistent nodular disease and no other recognized disease producing bronchiectasis, however, we believe the absence of long-term serial radiographic and clinical studies leaves the door open to the possibility that the bronchiectasis is a consequence rather than a cause of many of the patients with both diseases. A recently submitted abstract by Kuze and colleagues2 looked at serial computed tomography of pulmonary infections caused by MAC in patients without predisposing conditions. They noted that clusters of small peripheral nodules, with normal bronchi, was the initial radiographic abnormality, which progressed to bronchial thickening of the associated bronchi, and ultimately to cystic bronchiectasis. The latter change was seen a mean of 11.3 years after the onset of symptoms. This study suggests that bronchiectasis is a part of the disease process of MAC, at least in some patients. Again, more detailed studies of patients who present with lingular and middle lobe syndromes with assessment for associated nodular disease, careful evaluation for mycobacterial infection, and serial follow-up are needed to resolve satisfactorily this question of the relationship of bronchiectasis (cause or consequence) to MAC.

The final theory presented by Reich and Johnson is that of voluntary cough suppression as a cause of the middle lobe-lingular bronchiectasis and subsequent associated MAC disease. In our opinion, voluntary cough suppression is not sufficiently well described, or accepted as a definable entity, to attribute any pulmonary disease to this phenomenon.3,4 We would accept this as one of a number of potential possibilities to explain the bronchiectasis and MAC disease occurring in women. Genetic and age-related immunologic factors, unknown environmental risks, etc, should also be considered as potential factors that may be responsible for the disease. Additional clinical, pathologic, and epidemiologic studies of this "silent" epidemic are needed to turn theories into fact. Women with disease, it is your turn to stand up and be counted, and counted correctly.

Richard J. Wallace Jr., MD, FCCP, and David E. Griffith, MD, FCCP,
University of Texas Health Center, Tyler, Texas

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Producing Red Herring

To the Editor:

Jackson et al1 in the June issue of Chest confused Murphy’s Law (“What can go wrong, will go wrong.”) with Sutton’s Law (“Go where the money is.”) in their article “Left Upper Lobe Mass and Diffuse Reticular-Nodular Infiltrate.”

As I grew up in Brooklyn, New York in the 1940s and 1950s, the bank robbing exploits of Willie “the actor” Sutton were as memorable as his remarks soon after capture. When asked why he always robbed banks, he replied, “That’s where the money is.”

As a medical student in the early 1960s at SUNY Downstate Medical Center, I was privileged to work at the Cardiology Service of the legendary professor of medicine and former chairman of the department, William Dock, who first applied that aphorism (“That’s where the money is.”) to the practice of clinical medicine. He admonished students to use the single test most likely to bear fruit before undertaking a series of studies that do little more than produce red herrings.2

Kenneth M. Frankel, MD, FCCP, Thoracic Surgical Service, Baystate Medical Center, Springfield, Massachusetts

REFERENCES


Hospital-Acquired Morbidity and Circadian Rhythms in Patients in the ICU

To the Editor:

A recent article by Meyer and colleagues1 published in Chest have shown effects of adverse environmental factors in the ICU. We not only concur with their findings in the Journal but also we would like to emphasize that this is a significant factor in hospital-acquired morbidity (HAM) and remains unappreciated by the profession. We similarly reported ICU psychoses, especially common in postoperative elderly patients, have direct relevance to acute disturbance of circadian rhythms.2,4 Adverse environmental factors in the ICU should be part of ICU committee agenda and should reflect the level of quality of care and standards. Unfortunately, most ICU committees suffer from Lister-Koch syndrome, and even discussion of adverse environmental factors are considered “flaky.” Drs. Lister and Koch were giants of Victorian science and their discoveries led to decreases in mortality due to infectious diseases. Prevention of nosocomial infections in the ICU is important, minute attention to aseptic techniques is well admired, but researching and studying other causes of morbidity in the ICU should not be ignored. It is high